

Visualization of Lipid Metabolism in the Zebrafish Intestine Reveals a Relationship between NPC1L1-Mediated Cholesterol Uptake and Dietary Fatty Acid

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This article has been retracted: please see Elsevier Policy on Article Withdrawal (<http://www.elsevier.com/locate/withdrawalpolicy>).

This article has been retracted at the request of the authors.

In our original Resource, we reported a regulatory link between NPC1L1-mediated cholesterol uptake and dietary fatty acid in zebrafish larvae. In the absence of dietary long-chain fatty acids (LCFA), larval enterocytes failed to internalize luminal BODIPY-cholesterol. We attributed this LCFA dependence to the cholesterol transport protein NPC1L1, as dietary LCFAs were shown to induce the translocation of transgenic human NPC1L1 from a perinuclear compartment to the intestinal brush border (supported by the data shown in Figure 5 of the Resource). When overexpressed, human NPC1L1 localized to the brush border, where it was able to mediate the internalization of BODIPY-cholesterol even in the absence of dietary LCFAs (Figure 5). We also reported that NPC1L1 directly mediates cholesterol uptake by larval enterocytes (shown in Figure 6 of the Resource). However, we recently discovered that the larval expression of the human NPC1L1 transgenic construct *hsp70:HsNPC1L1-mCherry* we reported was in fact that of *hsp70:mCherryCAAX*, which encodes an mCherry fluorophore modified with a prenylation motif. When employing a recloned *hsp70:HsNPC1L1-mCherry* vector, our experiments failed to replicate the LCFA-induced translocation of NPC1L1 to the intestinal brush border, indicating that the findings reported in Figures 5 and 6 are no longer valid.

Our conclusions regarding zebrafish intestinal lipid droplet formation and depletion when given a high-fat meal, BODIPY-fatty acid incorporation into LDs, and BODIPY-cholesterol localization to the endocytic compartment of enterocytes, distinct from LDs, are not affected by this mistake. Additionally, the finding that dietary cholesterol absorption is dependent on luminal long-chain fatty acids is also unaffected. All of these experiments (Figures 1–4 in the Resource) were performed using zebrafish lines that did not contain the incorrect construct mentioned above.

Nonetheless, given that some of the core mechanistic conclusions we presented are no longer valid due to the use of the wrong construct, we are now retracting the paper. We apologize to the community if our error caused any significant confusion or inconvenience.